Coronary Cineangiography

Clinical Studies for Evaluation In Equivocal Cases

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■ *Coronary cineangiography was helpful in the following circumstances:*

1. A patient with an abnormal electrocardiogram and no evidence of coronary artery disease.

2. A patient with thoracic pain in whom the diagnosis of coronary artery disease was questioned.

3. A patient with intractable angina pectoris who was being studied for possible surgical therapy.

4. A patient with coronary insufficiency and aortic stenosis who was being evaluated for operation on the aortic valve.

THERE has been increasing interest in the visualization of arteries to demonstrate occlusive disease. With the development of newer techniques, opacification of the coronary arteries has been accomplished with minimal morbidity and mortality and consistently satisfactory results.

We use the Sones technique[4] for coronary angiography, the major modification being that the catheter is inserted percutaneously into the axillary artery through a needle puncture.[1] A 6-inch image intensifier with a 35 mm camera capable of 60 frames per second is used for catheter placement and recording the image. Cineflor film developed in Eastman's rapid fix solution has proven satisfactory for film processing.

In the average patient, the right axillary artery is punctured under sterile conditions and local anesthesia in the lower axilla. A guide wire of Seldinger type (1 cm outside diameter) 135 cm long is inserted into the artery and maneuvered under fluoroscopic control into the ascending aorta. The guide wire may be made to pass obstacles and smaller blood vessels by maneuvering the patient's arm and neck. Once the tip of the guide wire is in the ascending aorta, the needle is taken off at the proximal end of the guide wire. A No. 7 Sones catheter is passed over the guide wire and down to the skin. Both guide wire and catheter are inserted through the skin and into the artery. Under fluoroscopic control, with an assistant slowly withdrawing the guide wire, the Sones catheter is made to pass over the wire into the ascending aorta. By teamwork, the tip of the Sones catheter is made to replace the tip of the guide wire in position in the ascending aorta.

The catheter is attached to the three-way stop-

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cock system of Sones. Once a coronary artery has been catheterized, 3 to 4 cc injections of 75 per cent Hypaque® are made while cinefluorography is being carried out. Care is taken that the catheter tip does not totally occlude a coronary artery. The pressure at the catheter tip is constantly monitored with a transducer. Sufficient numbers of angiograms are taken to visualize all vessels without overlap of images. This usually requires at least three views of the left and two of the right coronary artery. The total volume of contrast material used in the average patient for finding the individual coronary arteries and the angiography averages about 125 cc.

The purpose of this paper is to illustrate with case reports some of the clinical uses of coronary cineangiography.

Report of Cases

CASE 1. The patient, a 27-year-old man, was first seen on October 18, 1963, because of nonspecific abnormalities in the T-wave noted on a routine electrocardiogram—inversion of the T-wave in leads 2, 3, AVF, and V-3 to V-6 unaltered by body position or hyperventilation. There was no history of cardiovascular disease or disability. In addition to his usual occupation, the patient was a semi-professional baseball player. Results of physical examination were within normal limits, as were blood cell count, urinalysis, protein-bound iodine and x-ray studies of the chest. The patient was walked on a treadmill at a 10 degree angle for ten minutes at four miles an hour, almost to the point of exhaustion, without distress in the chest or changes in the electrocardiogram change. However, the patient had been "rated up" for life insurance, restricted to sedentary activities, and told that playing baseball was hazardous for him. As a result he was somewhat anxious about himself. Therefore, coronary angiography was recommended. On November 14, 1963, coronary angiography revealed no evidence of occlusive disease. At last report the patient was playing baseball, was working at his job and was no longer apprehensive.

Discussion. We do not know the cause of the patient's abnormal electrocardiogram. Perhaps it is attributable to undiagnosed myocarditis in childhood. However, it is unwise to equate abnormalities in the electrocardiogram with coronary artery disease. In this case it was important from a personal and an economic standpoint to determine that the vascular tree was normal.

CASE 2. The patient, a 42-year-old man, was well until about January 1962 when he noted the sudden onset of sharp pain in the left inframammary area radiating to the inner aspect of the left arm. It was non-pleuritic, not related to body position and was associated with increased sweating and dyspnea. The patient was put in hospital for four weeks with a diagnosis of acute myocardial infarction. He returned to his job, a sedentary one, in May, 1962. The pains became progressively worse, lasting two hours at a time, occurring almost daily. Although at first he noted some relief from the use of sublingual nitroglycerine, by mid-year this drug was no longer effective. He stopped working in October 1962 because of the severity of the pains. He was put in hospital for seven days in April 1963 and was again told that he had an infarct. First seen in consultation on August 13, 1963, the patient said the pains were never related to physical exertion. Commonly they followed emotional liability or a period of leaning over. Nausea, but not vomiting, infrequently accompanied the pain. The past history was non-contributory. There were no significant findings on physical examination. Results of all laboratory tests were within normal limits, including determination of cholesterol, fasting blood sugar, serum transaminase (SGOT), creatinine, urine catecholamines, serum calcium and protein-bound iodine. A resting electrocardiogram was normal. No abnormalities were brought on by exercise tests. An electrocardiogram taken during pain was within normal limits. The patient was exercised on a treadmill at a 10 degree angle at four miles per hour for six minutes without change in the electrocardiogram. Review of the old hospital records showed no evidence of myocardial infarction by electrocardiogram or laboratory tests. Because of the record of hospitalization and the diagnosis of coronary artery disease, concurred in by cardiac consultants, it was felt that visualization of the coronary arteries was needed. On August 29, 1963, a coronary angiogram showed normal coronary arteries. X-ray studies of the lower gastrointestinal tract, the gallbladder and the spine showed no abnormalities. Result of a provocative test for esophagitis with dilute hydrochloric acid was equivocal. An upper gastrointestinal series revealed a sliding hiatal hernia with antral gastritis. A medical regimen for hiatal hernia was prescribed, the patient was reassured about his heart and he returned to work with almost complete relief of symptoms.

Discussion. Coronary angiography was desirable in this case because of the diagnosis of coronary disease made in the past. Careful attention to details of the history may have led one to suspect the correct diagnosis.[2]

CASE 3. A 48-year-old man gave a history of acute myocardial infarction in November 1957

with progressive intractable angina pectoris since that time. At the time of examination in September, 1963, the patient was taking 20 nitroglycerine tablets per day for substernal oppression, apparently precipitated by emotions, by minimal exertion such as walking across the room, and by assuming the recumbent position. He obtained relief from the nitroglycerine in 10 to 15 minutes. He was forced to stop working as a florist about September, 1963. With the resultant relative inactivity the pains were less frequent but still incapacitating. During the course of evaluation various agents for relief of the symptoms were given without any apparent effect. The only significant finding on physical examination was a grade III low pitched holosystolic murmur at the apex with axillary transmission. This murmur was thought not to have been present before the infarction. All laboratory tests including determination of protein-bound iodine were within normal limits as were roentgenograms of the stomach, colon, gallbladder and cervical and thoracic spine. Because of the apparent refractoriness of the angina pectoris, visualization of the coronary arteries was thought to be desirable to see if surgical operation might help. A coronary angiogram demonstrated diffuse coronary artery disease with multiple areas of narrowing. It was thought that the lesions were too extensive for surgical therapy.[3] At last report the patient was being treated with a beta adrenergic blocking agent with some benefit.

Discussion. The coronary angiogram in this patient established the presence and extent of occlusive disease and incidentally demonstrated the presence of mitral insufficiency (papillary muscle dysfunction perhaps). The condition was discussed with the patient and conservative methods of treatment were utilized.

CASE 4. A 55-year-old woman had rheumatic fever at 8 years of age, known valvular disease at 13 and progressive dyspnea for three or four years before her first examination by the authors, in 1962. At that time, she had dyspnea upon walking across the room and had nocturnal dyspnea two to three times a week, without symptoms of dizziness or syncope. For two to three years, she had "pressing" pains in the anterior chest that occurred one or two times a day, usually precipitated by exertion such as walking five blocks on the level or doing housework, and commonly associated with nocturnal dyspnea. The pains often lasted one to two hours and were alleviated but not eliminated by taking nitroglycerine. Most of the pertinent physical findings were confined to the heart. The point of maximal impulse was 2 cm inside the left anterior axillary line. A systolic thrill was palpated over the aortic area. A grade VI low pitched, ejection systolic murmur was maximally audible over the aortic area and was transmitted over the entire precordium and posterior thorax. A grade III early diastolic blowing murmur was heard over the left precordium. The blood pressure was 124/70 mm of mercury. An electrocardiogram showed left ventricular hypertrophy and strain. X-ray films of the chest showed left ventricular enlargement and aortic valve calcifications. Further evaluation was thought to be necessary to see if the thoracic pain was caused by coronary artery disease, by involvement of the coronary ostia with the aortic stenosis or by a combination of these conditions. A coronary angiogram demonstrated patent coronary arteries with hypertrophy of both the right and circumflex branch of the left coronary arteries.

Discussion

In our experience with clinical-pathological correlations, the veracity of the angiograms has been excellent. From observation we have come to the belief that if there is no evidence of obstructive disease in these studies, the patient almost certainly has no clinical coronary artery disease. Conversely, it is possible to have thickening of the coronary arteries yet a normal angiogram, but we believe that these thickenings are not sufficient to cause clinical coronary artery disease. A normal angiogram is not equivalent to normal coronary arteries but would almost certainly eliminate suspicion of clinical coronary artery disease of a type to produce myocardial infarction.

Determining the status of the coronary arteries in patients with aortic stenosis and thoracic pain apparently due to coronary insufficiency is of more than academic interest. This information is of value in determining the desirability of operation on the aortic valve, in following progress of the patient through the surgical procedure and in anticipating changes in the clinical status postoperatively.

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